

Structure of internalising symptoms in early adulthood[†]

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Background Debate surrounds the underlying structure of internalising disorders including major depression, generalised anxiety disorder, phobias and panic disorders.

Aims To model the within-time and across-time relationships of internalising symptoms, incorporating effects from generalised internalising and disorder-specific components of continuity.

Method Data were gathered from a 25-year longitudinal study of a birth cohort of 953 New Zealand children. Outcome measures included DSM–IV symptom scores for major depression, generalised anxiety disorder, phobia and panic disorder at the ages of 18, 21 and 25 years.

Results Structural equation modelling showed that, within-times, a common underlying measure of generalised internalising explained symptom score comorbidities. Across-time correlation of symptom scores was primarily accounted for by continuity over time in generalised internalising. However, for major depression and phobia there was also evidence of across-time continuity in the disorder-specific components of symptoms.

Conclusions Internalising symptoms can be partitioned into components reflecting both a generalised tendency to internalising and disorder-specific components.

Declaration of interest None.

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There has been a large amount of research, debate and speculation about the classification of internalising disorders including major depression, generalised anxiety disorder, phobia and panic disorders (Kovacs & Devlin, 1998; Zahn-Waxler *et al.*, 2000; Lilienfeld, 2003; Watson, 2005). A central issue in these debates has concerned the extent to which internalising disorders are reflections of a common underlying disorder of internalising and the extent to which these disorders are distinct diagnostic entities (Kendler *et al.*, 1992; Brown *et al.*, 1998; Krueger *et al.*, 1998; Krueger, 1999; Hartman *et al.*, 2001; Vollebergh *et al.*, 2001; Hudson *et al.*, 2003; Hettema *et al.*, 2004; Kendler, 2004; Khan *et al.*, 2005; Schoevers *et al.*, 2005; Watson, 2005). Resolution of this issue is central to both the development of methods for classifying internalising disorders and for understanding the aetiological processes that underlie these disorders (Kendler *et al.*, 1992; Brown *et al.*, 1998; Kovacs & Devlin, 1998; Krueger *et al.*, 1998; Krueger, 1999; Hudson *et al.*, 2003; Lilienfeld, 2003; Hettema *et al.*, 2004; Clark, 2005).

In this paper we develop a structural equation model of the underlying structure of internalising disorder symptoms, and we fit this model to data gathered on a birth cohort of nearly 1000 young people studied on three occasions from the ages of 18 to 25 years. The general aims of this model were to examine the role of generalised and disorder-specific factors in the within-time comorbidity of disorder and the across-time continuity of disorders. Underlying this model is a general concern with estimating the fractions of variance and covariance between internalising symptoms which can be explained by a generalised tendency to

internalising, and how much of this variance and covariance is disorder-specific.

METHOD

Background to the model

Fig. 1 shows a conceptual model of the within- and across-time structures of internalising symptoms (major depression, generalised anxiety disorder, phobia, panic) assessed at three time periods (t1, t2, t3). The structure of the model can be thought of as comprising two linked components.

- The within-time model assumes that the observed symptom measures of major depression, generalised anxiety disorder, phobia and panic at each time *t* are linked by a common factor model in which the variance in the symptom scores reflects variation due to a generalised internalising factor (*I_t*), and variation specific to each disorder (*U_{it}*).
- The across-time model assumes that continuities between disorders can arise by two routes: mediated via the continuity of the generalised internalising factor across time, and disorder-specific.

If it is assumed that all relationships within the model are linear and additive, then the model in Fig. 1 may be written as a structural equation model. The full specification of the model is given in the statistical section below.

The major advantage of the conceptual model in Fig. 1 is that it resolves the lumpers/splitters debate by partitioning the variance of the symptom scores into components reflecting generalised internalising and disorder-specific variance. Further, the across-time model makes it possible to examine the extent to which continuities in internalising symptoms are mediated by the across-time stability of generalised internalising or via disorder-specific pathways. Finally, the model has the advantage of being testable, since the number of model parameters is smaller than the number of observed variances and covariances (see discussion of the identification status of the model below).

In the remainder of this paper we will fit the model in Fig. 1 to data on DSM–IV (American Psychiatric Association, 1994) internalising symptoms gathered on a birth cohort of young adults studied at ages 18, 21 and 25 years. The aims of this analysis are to determine the extent to which the

[†]See editorial, pp. 481–483, this issue.

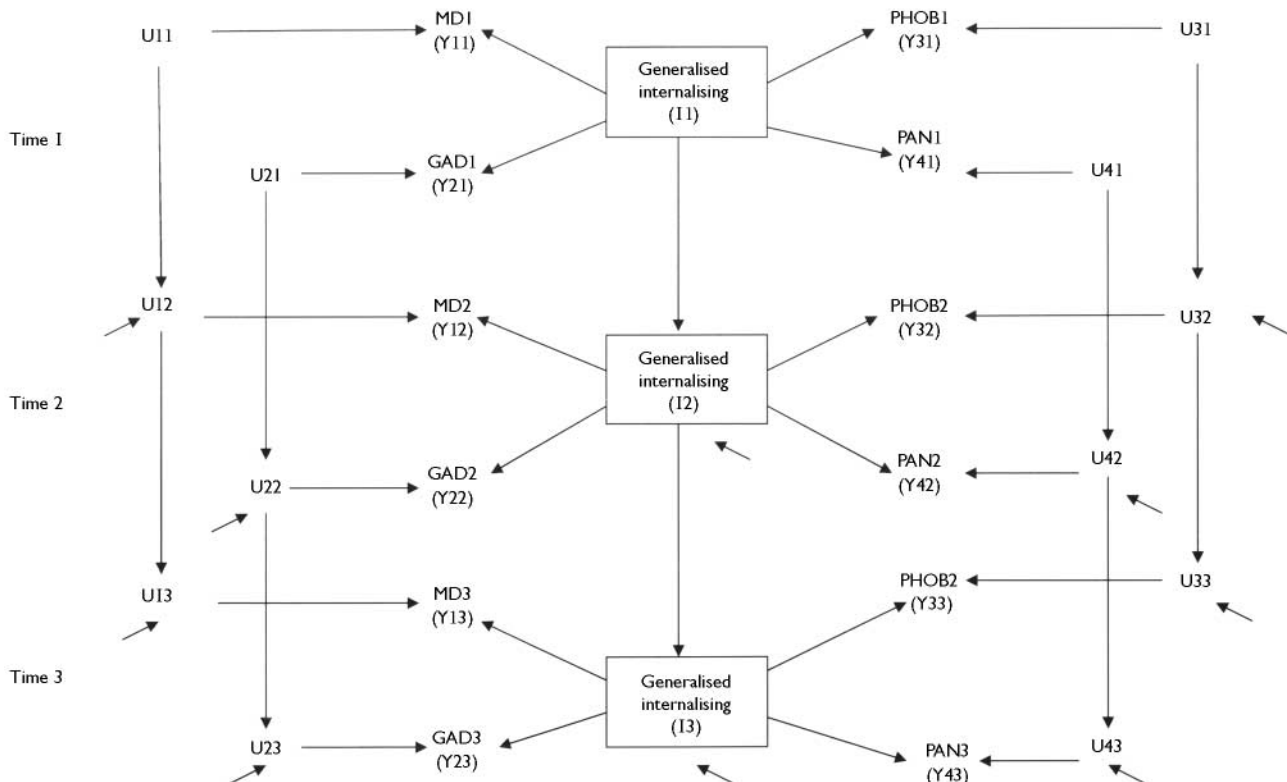


Fig. 1 Hypothetical model of within-time and across-time structure of internalising symptoms at three times. MDt, major depression symptom score at time t; GADt, generalised anxiety symptom score at time t; PHOBt, phobia symptom score at time t; PANt, panic symptom score at time t; It, generalised internalising score at time t ($t=1, 2, 3$). A detailed description of the model specification is given in the statistical analysis section. In this specification the Y_{it} ($i=1, 2, 3, 4$; $t=1, 2, 3$) represents the observed symptom score for the i -th symptom type (MD, GAD, PHOB, PAN respectively) at time t , and U_{it} is the disorder-specific component of Y_{it} that is not in common with generalised internalising.

model in Fig. 1 provides an adequate account of within- and across-time relationships between internalising disorder symptoms, and to examine the implications of the model for diagnostic classification and the understanding of the origins of internalising disorders.

Participants

The data were gathered during the course of the Christchurch Health and Development Study. In this study a birth cohort of 1265 children (635 boys and 630 girls, born in the Christchurch, New Zealand urban region in mid-1977) was studied at birth, 4 months, 1 year, annually to 16 years, and at 18, 21 and 25 years (Fergusson *et al.*, 1989; Fergusson & Horwood, 2001). The present analyses are based on the sample of 953 study participants who were interviewed on measures of internalising disorders at the ages of 18, 21 and 25 years. This sample represented 75% of the initial cohort of participants enrolled in the study. All study information was collected on the basis

of signed and informed consent from participants.

Internalising symptoms

Study participants were interviewed at the ages of 18, 21 and 25 years on a structured mental health interview designed to assess aspects of mental health and psychosocial adjustment since the previous assessment. All interviews were conducted in private by trained lay interviewers at a location convenient to the respondent. As part of the mental health assessment at each age, components of the Composite International Diagnostic Interview (CIDI; World Health Organization, 1993) were used to assess DSM-IV symptom criteria for a range of internalising disorders, including major depression, general anxiety disorder, social phobia, specific phobia, and panic disorders with or without agoraphobia. Using these data, summary measures of the extent of internalising disorder symptoms were constructed for each of the periods 16–18 years, 18–21 years and 21–25 years in the following ways.

Major depression

At each interview, participants were questioned about major depressive symptoms occurring in the past month, the past 12 months and the period back to the previous assessment. Participants who at any time reported a depressive episode involving either of the two core symptom criteria for major depression (feeling sad, miserable or depressed, or loss of interest in daily activities) were further questioned about the occurrence of other DSM-IV symptoms. For the purposes of the present analysis, a depressive symptoms score was constructed for each assessment period based on a count of the number of DSM-IV major depression symptoms reported at any time during the assessment period.

Generalised anxiety disorder

At each interview, participants were questioned about the occurrence of episodes of feeling tense, anxious or worried most of the time since the previous assessment. Young people who reported an episode lasting at least 1 month or longer were further questioned about the duration and

source of the anxiety and associated DSM-IV criterial symptoms. For the purposes of the present analysis, a generalised anxiety disorder symptom score was constructed for each assessment period, based on a count of the number of anxiety symptoms reported from the following list of DSM-IV criterial symptoms: feeling restless, keyed up or on edge; getting tired very easily; having difficulty concentrating; feeling irritable; muscles feeling tense, sore or aching; having trouble getting to sleep or staying asleep.

Phobia

Participants were questioned about DSM-IV criterial symptoms for social and specific phobia, including the nature of the fear, the level of distress experienced, avoidant behaviours, the extent of impairment of functioning and the extent of anxiety symptom experienced upon exposure to the source. For the purposes of the present analysis, a phobia symptoms score was computed for each interview period, based on a count of the number of anxiety symptoms that the young person reported experiencing when exposed to any social or specific phobia stimulus. These symptoms included: feeling nervous and panicky; sweating; heart beating faster; shortness of breath; blushing or shaking; feeling like vomiting; concern that they might do something embarrassing.

Panic

At each interview, participants were questioned about panic attacks occurring since the previous assessment, and CIDI items were used to assess relevant DSM-IV criterial symptoms. As part of this questioning, participants were asked to describe their most serious panic attack occurring during the interview period and any associated symptoms. For the purposes of the present analysis, a panic symptoms score was created for each interview period, based on a count of the number of panic attack symptoms reported for the most severe attack out of the list of 13 DSM-IV criterial symptoms. In view of the low base rate of panic, no attempt was made to distinguish between panic attacks occurring in the presence or absence of agoraphobia.

Statistical analysis

The above measures of internalising, comprising four symptom scores (major

depression, generalised anxiety disorder, phobia, panic) assessed at three time periods, formed the input data for fitting the model depicted in Fig. 1. Let Y_{it} represent the symptom score for the i -th diagnostic domain ($i=1, 2, 3, 4$) at the t -th time period ($t=1, 2, 3$). It represent the measure of generalised internalising at each time, t and U_{it} the disorder-specific component of Y_{it} . Then, subject to the assumption that the associations between variables are linear and additive, this model may be represented as a structural equation model defined by the following system of equations.

Within-time model:

$$Y_{it} = \lambda_{it} It + U_{it} \quad i=1, 2, 3, 4 \quad t=1, 2, 3$$

Across-time model:

$$It = \gamma_t It_{-1} + Et \quad t=2, 3$$

$$U_{it} = Bit U_{it-1} + Wit \quad i=1, 2, 3, 4 \quad t=2, 3$$

In these equations, the coefficients λ_{it} represent the factor loadings of the observed symptom scores (Y_{it}) on the underlying measures of generalised internalising (It). If all variables in the model are standardised, the squares of these coefficients represent the proportion of variance in the observed symptom scores that is accounted for by generalised internalising. The across-time continuities in generalised internalising (It) and disorder-specific components (U_{it}) are assumed to be related by an autoregressive model with coefficients γ_t and Bit respectively. The terms Et and Wit represent disturbance terms in the across-time components of the model. These disturbance terms are assumed to be mutually uncorrelated. In addition, the model assumes that both the disorder-specific components U_{it} and the disturbances Wit are uncorrelated with the measures of generalised internalising It .

The above model may be fitted to the correlation matrix of the 12 observed symptom scores (4 disorder symptom scores at 3 times). A necessary condition for the model to be identifiable (estimable) is that the number of model parameters to be estimated is less than or equal to the number of non-redundant elements (k) of the observed correlation matrix ($k=78$). The model specification for Fig. 1 has a total of 34 parameters to be estimated (12 factor loadings λ_{it} , 8 parameters Bit , 2 parameters γ_t and 12 variances for the terms U_{it} and Wit). The model is identified with 44 degrees of freedom. Further, because the number of model parameters is substantially less than the number of

non-redundant correlation elements, the model is falsifiable to the extent that an inadequate model may be rejected on the basis of poor fit to the observed data.

In the present analysis, models were fitted to the matrix of polychoric correlations between the observed symptom measures. Model fitting was conducted using LISREL 8 (Joreskog & Sorbom, 1993a) and methods of weighted least squares estimation. These methods are more appropriate for the situation in which data are non-normally distributed (Joreskog & Sorbom, 1993a), and were used in the present instance because the observed report data were highly skewed. Assessment of model fit was based on evaluation of a number of fit indices including the chi-squared goodness-of-fit index, the root mean-squared error of approximation (RMSEA), the root mean-squared residual correlation (RMSR), the adjusted goodness-of-fit index (AGFI), and the comparative fit index (CFI). A well-fitting model should have an RMSEA of less than 0.05, an RMSR close to zero, and AGFI and CFI indices close to 1 (Joreskog & Sorbom, 1993b). Finally, the model was extended to include gender, and tests of gender heterogeneity were conducted using the multiple indicators, multiple causes (MIMIC) modelling approach described by Muthen (Muthen, 1989).

RESULTS

Correlations

Table 1 shows the matrix of polychoric correlations between the measures of major depression, generalised anxiety disorder, phobia and panic symptom scores assessed at ages 16–18, 18–21 and 21–25 years. This table shows the presence of significant correlations between measures both within and across time periods.

Model fitting

The conceptual model in Fig. 1 showed a generally good fit to the data in terms of measures of goodness-of-fit (RMSEA = 0.032, P -value for test of close fit (RMSEA < 0.05) = 0.99; RMSR = 0.065; AGFI = 0.98; CFI = 0.98). However, the model chi-square statistic proved to be significant ($\chi^2 = 85.5$; d.f. = 44; $P = 0.0002$). Examination of modification indices and model residuals suggested the model fit could be significantly improved by two changes to the original model specification.

Table 1 Matrix of polychoric correlations between major depression, generalised anxiety disorder, phobia and panic symptom scores (at 18, 21 and 25 years of age)

| Measure | Y11 | Y21 | Y31 | Y41 | Y12 | Y22 | Y32 | Y42 | Y13 | Y23 | Y33 | Y43 |
|------------------------|------|------|------|------|------|------|------|------|------|------|------|------|
| Age 18 years | | | | | | | | | | | | |
| Major depression (Y11) | 1.00 | | | | | | | | | | | |
| GAD (Y21) | 0.56 | 1.00 | | | | | | | | | | |
| Phobia (Y31) | 0.45 | 0.45 | 1.00 | | | | | | | | | |
| Panic (Y41) | 0.55 | 0.58 | 0.57 | 1.00 | | | | | | | | |
| Age 21 years | | | | | | | | | | | | |
| Major depression (Y12) | 0.51 | 0.29 | 0.30 | 0.36 | 1.00 | | | | | | | |
| GAD (Y22) | 0.41 | 0.41 | 0.28 | 0.47 | 0.51 | 1.00 | | | | | | |
| Phobia (Y32) | 0.33 | 0.26 | 0.51 | 0.36 | 0.35 | 0.31 | 1.00 | | | | | |
| Panic (Y42) | 0.38 | 0.38 | 0.37 | 0.49 | 0.49 | 0.60 | 0.60 | 1.00 | | | | |
| Age 25 years | | | | | | | | | | | | |
| Major depression (Y13) | 0.38 | 0.19 | 0.21 | 0.32 | 0.48 | 0.29 | 0.27 | 0.41 | 1.00 | | | |
| GAD (Y23) | 0.28 | 0.20 | 0.18 | 0.38 | 0.39 | 0.30 | 0.22 | 0.40 | 0.57 | 1.00 | | |
| Phobia (Y33) | 0.28 | 0.18 | 0.42 | 0.34 | 0.27 | 0.22 | 0.56 | 0.40 | 0.37 | 0.32 | 1.00 | |
| Panic (Y43) | 0.17 | 0.15 | 0.21 | 0.26 | 0.34 | 0.21 | 0.36 | 0.48 | 0.52 | 0.53 | 0.44 | 1.00 |

GAD, generalised anxiety disorder.

First, for major depression and phobia an additional disorder-specific pathway from time 1 (age 18 years) to time 3 (age 25 years) was included in the model. Second, the disorder-specific components of major depression and generalised anxiety disorder were permitted to be correlated within measurement periods.

These changes in model structure led to a significant improvement in model fit ($\Delta\chi^2=24.0$, d.f.=5, $P<0.001$) and produced an adequately fitting model on the basis of the fit indices (RMSEA=0.025, P -value for test of close fit (RMSEA < 0.05)=1.00; RMSR=0.056; AGFI=0.98; CFI=0.99). The final fitted model is shown in Fig. 2. The figure gives the standardised model parameters. For ease of presentation, non-significant ($P>0.05$) pathways and some disturbance terms have been omitted from the model. Examination of the figure shows the following.

Model interpretation

The within-time structure

The fitted model shows that each of the measures (major depression, generalised anxiety disorder, phobia and panic) had strong and statistically significant loadings on the generalised internalising factor. These loadings ranged from 0.64 to 0.89 with a median value of 0.66. This aspect

of the model makes it possible to decompose the variance of each test at each time into two uncorrelated components: the test variance that was in common with the generalised internalising measure, and the test variance specific to the measure. These variance decompositions are shown in Table 2. This table shows that the generalised internalising factor explained in the region of 41% to 45% of the variance in major depression symptoms; 42% to 50% of the variance in generalised anxiety disorder symptoms; 37% to 42% of the variance in phobia symptoms; and 62% to 78% of the variance in panic symptoms. These results suggest that the generalised internalising factor explained from just under a half to just over three-quarters of the observed symptom score variance, with the remaining variance (including error variance) being specific to the test. Furthermore, aside from the small correlation between the residuals for major depression and generalised anxiety disorder, all of the within-time comorbidity of the observed symptom scores was explained by generalised internalising.

The across-time structure

The fitted model shows that there were two general routes leading to the across-time continuity of symptom scores. First, this

continuity was mediated by the linkages between each test and the generalised internalising factor. Second, there was homotypic continuity independently of the mediating effect of generalised internalising. These features of the model make it possible to decompose the across-time correlations of tests into two additive components: the component mediated via generalised internalising, and the component independent of generalised internalising. These decompositions are given in Table 3. The table shows that all of the across-time continuity of generalised anxiety disorder and panic symptoms was mediated by generalised internalising. However, for major depression and phobia there was evidence of further pathways in which the presence of symptoms at one time influenced the same type of symptoms at a later time independently of the effects of generalised internalising. The results show that, for major depression symptoms, in the region of 62% to 69% of the across-time correlations were mediated via generalised internalising and the remainder were specific to depression. For phobia symptoms, between 56% and 58% of the across-time correlations were mediated via generalised internalising and the remainder were specific to phobia.

Supplementary analysis

To examine the extent to which the core model structure varied with gender, the model fitted in Fig. 2 was extended to include gender, and the methods described by Muthén (Muthén, 1989) were used to test for gender heterogeneity. This analysis showed that gender was significantly correlated with the measures of generalised internalising ($r=0.28$ to 0.47 , $P<0.001$), reflecting a significant tendency for girls and women to exhibit higher general levels of internalising behaviour. However, there was no evidence to suggest that other aspects of model structure, including the factor loadings for the internalising symptom scores and the continuities of either the generalised internalising or the specific disorder components, varied with gender.

DISCUSSION

The structure of internalising

In this paper we have used data gathered over the course of a longitudinal study to examine the within- and across-time structure of DSM-IV symptom measures of

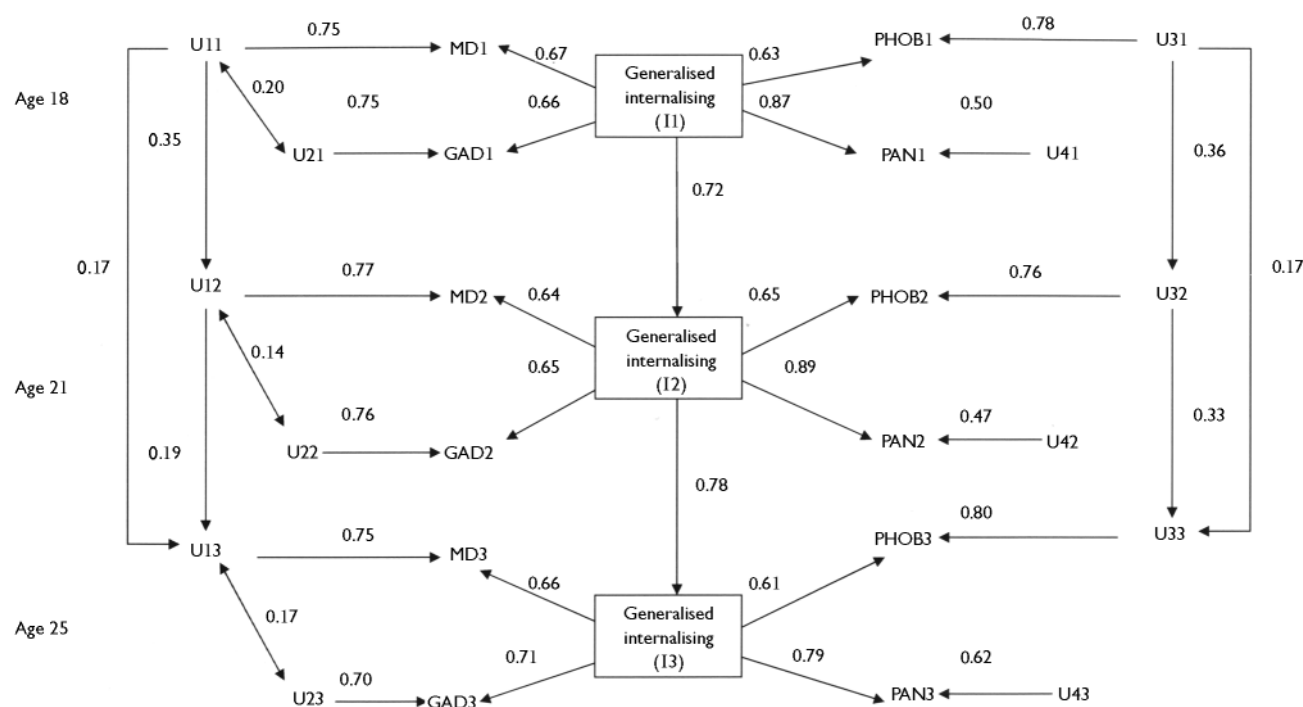


Fig. 2 Fitted model of internalising symptoms at ages 18, 21, and 25 years. MDt, major depression symptom score at time t; GADt, generalised anxiety symptom score at time t; PHOBt, phobia symptom score at time t; PANt, panic symptom score at time t; It, generalised internalising score at time t (t=1, 2, 3). Uit (i=1, 2, 3, 4; t=1, 2, 3) represents the disorder-specific component of the observed symptom score for the i-th symptom type (MD, GAD, PHOB, PAN respectively) at time t.

internalising disorders including major depression, generalised anxiety disorder, phobias and panic disorders. The best fitting structural model proved to be a hybrid of two traditions that have dominated the description of internalising symptoms. First, the results support the view that the current DSM disorder classifications represent unique and to some extent non-overlapping domains of internalising behaviours. At the same time, the model also suggests that the within-time comorbidity of these domains of disorder is explained by a common generalised internalising factor. Thus, there is evidence for both the lumpers and splitters positions on the classification of internalising disorders, with some fraction of the variance in symptom scores reflecting a generalised dimension of internalising, whereas the

remaining variance is disorder-specific. These conclusions are generally consistent with the results of previous models of symptom data that have produced evidence for both lumpers and splitters positions (Mineka *et al*, 1998; Krueger, 1999, 2002; Watson, 2005).

On occasions, the diagnostic categories reported in nosologies such as DSM-IV are treated as if they represent homogeneous disorders having a common set of aetiological factors. The present analysis suggests that such an interpretation is implausible, in that the origins of these disorders are likely to be complex and heterogeneous, reflecting factors that are common to all internalising and factors that are specific to a given disorder. The model estimates suggest that in the region of half to three-quarters of the variance

in disorder symptom scores reflects a generalised internalising factor, with the remaining variance being specific to the specific disorders.

Across-time continuities

It has been well documented that internalising disorders tend to recur, and there is evidence of both homotypic continuity in which the same disorders show recurrence over time, and heterotypic continuity in which the onset of one disorder leads to an increased risk of the later onset of other internalising disorders (Keller *et al*, 1992a,b; McGee *et al*, 1992; Angold *et al*, 1999; Lilienfeld, 2003). An understanding of the development of internalising disorders thus requires models that take into account both homotypic and heterotypic continuity. The model developed in this paper achieves this by permitting continuity of disorder by two routes. First, it is assumed that continuity of disorder may arise via the continuity of the generalised internalising factor across time. Second, the model permits specific homotypic continuity of disorders.

The fitted model leads to two major conclusions about the nature of across-time continuity in internalising disorders. First, much of the across-time continuity in

Table 2 Percentage of within-time variance in disorder symptom scores owing to generalised internalising

| Symptom measure | % of variance owing to internalising | | |
|------------------|--------------------------------------|-----------------|-----------------|
| | 18 years of age | 21 years of age | 25 years of age |
| Major depression | 44.5 | 41.3 | 43.6 |
| GAD | 43.4 | 41.7 | 50.4 |
| Phobia | 39.3 | 41.6 | 36.8 |
| Panic | 75.0 | 78.3 | 61.8 |

GAD, generalised anxiety disorder.

Table 3 Decomposition of across-time correlations of disorder symptom scores

| Correlation | Estimated total correlation from fitted model | Component owing to continuity in internalising | Component owing to disorder-specific continuity |
|-------------------------|---|--|---|
| Major depression | | | |
| Age 18–21 | 0.51 | 0.31 | 0.20 |
| Age 21–25 | 0.48 | 0.33 | 0.15 |
| Age 18–25 | 0.38 | 0.25 | 0.13 |
| GAD | | | |
| Age 18–21 | 0.39 | 0.39 | NS |
| Age 21–25 | 0.35 | 0.35 | NS |
| Age 18–25 | 0.26 | 0.26 | NS |
| Phobia | | | |
| Age 18–21 | 0.50 | 0.29 | 0.21 |
| Age 21–25 | 0.54 | 0.31 | 0.23 |
| Age 18–25 | 0.39 | 0.22 | 0.17 |
| Panic | | | |
| Age 18–21 | 0.45 | 0.45 | NS |
| Age 21–25 | 0.52 | 0.52 | NS |
| Age 18–25 | 0.39 | 0.39 | NS |

GAD, generalised anxiety disorder; NS, disorder-specific pathway not significantly different from zero.

internalising disorders reflects the strong across-time continuity of the internalising factors. This result suggests that much of the homotypic and all of the heterotypic continuity in internalising disorders arises because individuals predisposed to high levels of internalising show the recurrence of the same disorders and the onset of new disorders. At the same time, the results make it clear that not all of the across-time continuity in internalising disorders is mediated via generalised internalising, and there is evidence of disorder-specific homotypic continuity, this being most marked for major depression and phobias. In this respect the findings for across-time continuity mirror the findings for within-time comorbidity, and suggest the presence of both a generalised internalising component and disorder-specific components.

Generalised internalising

In turn, these findings raise speculations about the interpretation of the generalised internalising factors postulated in this analysis. This factor can be interpreted in at least three ways. First, it may be suggested that this factor represents variation in individual predisposition to internalising disorders. Under this interpretation, the generalised internalising factor has a similar interpretation to the personality trait of

neuroticism (Eysenck, 1990). As a number of authors have pointed out, the trait of neuroticism may largely or wholly reflect individual variation in stable levels of internalising symptoms (Duncan-Jones *et al*, 1990; Ormel *et al*, 2004). Second, it is possible that the internalising factor does not represent a dimension of personality or disorder, but rather is a latent variable that summarises the net effects of non-observed genetic and environmental factors on individual tendencies to internalising symptoms. Finally, the internalising factor could be conceptualised as an underlying dimension reflecting the extent of generalised internalising disorder. This conceptualisation would support the view that there may be value in extending current systems of diagnostic classification to include a category of generalised internalising disorder. At the present time, there is no evidence to determine which of these interpretations is the more correct. None the less, what the analysis does make clear is that there is considerable overlap, correlation and comorbidity between internalising disorders, with this overlap adequately represented by a single, general and highly stable latent dimension.

Limitations

There are a number of important caveats that need to be placed on these results. First

and foremost, the findings describe the within- and across-time structures of internalising disorders in a specific cohort, studied at a specific life stage using a specific set of measures. The extent to which the findings generalise beyond this context remains to be explored. A second potential limitation of the analysis is that we have assumed that the current DSM-IV groupings of internalising symptoms into major depression, generalised anxiety disorder, phobias, and panic disorders provides a valid account of symptom variation. Further, to secure sufficient variation for analysis we have combined some disorders (notably phobias). These coding and classification rules may influence the results and conclusions drawn.

Despite these limitations, the model developed in this paper has the major advantage that it provides a resolution to the long-standing lumpers/splitters debate by showing that variation in internalising symptoms can be partitioned into generalised and disorder-specific components, with this dissection being evident in both within-time analyses of comorbidity and across-time analyses of continuity.

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REFERENCES

- American Psychiatric Association (1994) *Diagnostic and Statistical Manual of Mental Disorders* (4th edn) (DSM-IV). Washington, DC: APA.
- Angold, A., Costello, E. & Erkanli, A. (1999) Comorbidity. *Journal of Child Psychology and Psychiatry*, **40**, 57–87.
- Brown, T. A., Chorpita, B. F. & Barlow, D. H. (1998) Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology*, **107**, 179–192.
- Clark, L. A. (2005) Temperament as a unifying basis for personality and psychopathology. *Journal of Abnormal Psychology*, **114**, 505–521.
- Duncan-Jones, P., Fergusson, D. M., Ormel, J., *et al* (1990) A model of stability and change in minor psychiatric symptoms: results from three longitudinal studies. *Psychological Medicine*, **18**, 1–28.
- Eysenck, H. J. (1990) Genetic and environmental contributions to individual differences: the three major dimensions of personality. *Journal of Personality*, **58**, 245–261.
- Fergusson, D. M. & Horwood, L. J. (2001) The Christchurch Health and Development Study: review of

findings on child and adolescent mental health. *Australian and New Zealand Journal of Psychiatry*, **35**, 287–296.

Fergusson, D. M., Horwood, L. J., Shannon, F. T., et al (1989) The Christchurch Child Development Study: a review of epidemiological findings. *Paediatric and Perinatal Epidemiology*, **3**, 278–301.

Hartman, C., Hox, J., Mellenbergh, G. J., et al (2001) DSM–IV internal construct validity: when a taxonomy meets data. *Journal of Child Psychology and Psychiatry*, **42**, 817–836.

Hettema, J. M., Prescott, C. A. & Kendler, K. S. (2004) Genetic and environmental sources of covariation between generalized anxiety disorder and neuroticism. *American Journal of Psychiatry*, **161**, 1581–1587.

Hudson, J. I., Mangweth, B., Pope, H. G., Jr, et al (2003) Family study of affective spectrum disorder. *Archives of General Psychiatry*, **60**, 170–177.

Joreskog, K. G. & Sorbom, D. (1993a) *Lisrel 8 User's Reference Guide*. Chicago, IL: Scientific Software International.

Joreskog, K. G. & Sorbom, D. (1993b) *LISREL 8: Structural Equation Modelling with the SIMPLIS Command Language*. Chicago, IL: Scientific Software International.

Keller, M. B., Lavori, P. W., Mueller, T. I., et al (1992a) Time to recovery, chronicity, and levels of psychopathology in major depression: a 5-year prospective follow-up of 431 subjects. *Archives of General Psychiatry*, **49**, 809–816.

Keller M. B., Lavori, P. W., Wunder, J., et al (1992b) Chronic course of anxiety disorders in children and adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, **31**, 595–599.

Kendler, K. S. (2004) Major depression and generalized anxiety disorder: same genes, (partly) different environments – revisited. *Focus*, **2**, 416–425.

Kendler, K. S., Neale, M. C., Kessler, R. C., et al (1992) Major depression and generalized anxiety

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disorder. Same genes, (partly) different environments? *Archives of General Psychiatry*, **49**, 716–722.

Khan, A. A., Jacobson, K. C., Gardner, C. O., et al (2005) Personality and comorbidity of common psychiatric disorders. *British Journal of Psychiatry*, **186**, 190–196.

Kovacs, M. & Devlin, B. (1998) Internalizing disorders in childhood. *Journal of Child Psychology and Psychiatry*, **39**, 47–63.

Krueger, R. F. (1999) The structure of common mental disorders. *Archives of General Psychiatry*, **56**, 921–926.

Krueger, R. F. (2002) The structure of common mental disorders. Reply. *Archives of General Psychiatry*, **59**, 570–571.

Krueger, R. F., Caspi, A., Moffitt, T. E., et al (1998) The structure and stability of common mental disorders (DSM–III–R): a longitudinal–epidemiological study. *Journal of Abnormal Psychology*, **107**, 216–227.

Lilienfeld, S. O. (2003) Comorbidity between and within childhood externalizing and internalizing disorders: reflections and directions. *Journal of Abnormal Child Psychology*, **31**, 285–291.

McGee, R., Feehan, M., Williams, S., et al (1992) DSM–III disorders from age 11 to age 15 years. *Journal of the American Academy of Child and Adolescent Psychiatry*, **31**, 50–59.

Mineka, S., Watson, D. & Clark, L. A. (1998) Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology*, **49**, 377–412.

Muthen, B. (1989) Latent variable modelling in heterogeneous populations. *Psychometrika*, **54**, 557–585.

Ormel, J., Rosmalen, J. & Farmer, A. (2004) Neuroticism: a non-informative marker of vulnerability to psychopathology. *Social Psychiatry and Psychiatric Epidemiology*, **39**, 906–912.

Schoevers, R. A., Deeg, D. J. H., van Tilburg, W., et al (2005) Depression and generalized anxiety disorder. *American Journal of Geriatric Psychiatry*, **13**, 31–39.

Vollebergh, W. A. M., Iedema, J., Bijl, R. V., et al (2001) The structure and stability of common mental disorders. *Archives of General Psychiatry*, **58**, 597–603.

Watson, D. (2005) Rethinking the mood and anxiety disorders: a quantitative hierarchical model for DSM–IV. *Journal of Abnormal Psychology*, **114**, 522–536.

World Health Organization (1993) *Composite International Diagnostic Interview (CIDI)*. Geneva: WHO.

Zahn-Waxler, C., Klimes-Dougan, B. & Slaterry, M. J. (2000) Internalizing problems of childhood and adolescence: prospects, pitfalls, and progress in understanding the development of anxiety and depression. *Development and Psychopathology*, **12**, 443–466.

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